Being overweight in life, not just childhood, increases risk of heart disease and diabetes

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The association between childhood obesity and increased risk of coronary artery disease and type 2 diabetes is due to individual's remaining overweight into adulthood, finds a new University of Bristol

study. The research, published in *The BMJ*, investigated how the genetic influence of being overweight in 453,169 individuals at different stages of life contributed towards their disease risk.

Obesity in childhood is known to have a detrimental impact on various health conditions and disease risk in later life including coronary heart disease, type 2 diabetes and cancer. However it is unclear whether being overweight as a child directly influences risk of these diseases or whether they can be reversed through lifestyle changes, particularly as those who are obese in early life tend to remain obese as adults.

Researchers from Bristol Medical School sought to investigate this by using a unique cause-and-effect analytical technique called Mendelian randomisation, which allows scientists to separate the genetic influence of risk factors, such as being overweight as a either a child or as an adult, on risk of disease—such as coronary artery disease, type 2 diabetes, breast and prostate cancer.

The technique was applied using human genetic data from 453,169 individuals from the UK Biobank study and four large scale genome-wide association studies using measures of BMI in adulthood (average age 57) and self-reported perceived body size at age 10.

The researchers found evidence that childhood obesity is associated with increased risk of coronary heart disease and type 2 diabetes due to a persistent, long-term effect of obesity over many years. This indicates that within a population, individuals who are overweight as children are more likely to be at risk of these diseases as they tend to remain overweight as adults. However, encouragingly this suggests that lowering weight in adulthood could reduce the long-term adverse effects of childhood obesity.

In contrast, their findings also provided evidence that having a smaller

body size during childhood might increase the risk of breast cancer regardless of body size in adulthood, with timing of puberty also likely playing a role, a finding which needs further research to understand its implications. While no strong evidence was found of a causal effect of either early or later life measures on prostate cancer, this disease should be revisited once data on a larger number of cases are available.

Dr. Tom Richardson, a UKRI Innovation Research Fellow in Genetic Epidemiology at Bristol Medical School's MRC Integrative Epidemiology Unit, explains: "Our findings for coronary heart disease and type 2 diabetes suggest that, if changes to genetically predicted early life equate to weight change through diet and exercise, then there exists a window of opportunity between childhood and adulthood to mitigate the effect of childhood obesity on disease risk. Our findings on breast cancer raise questions about the role that timing of puberty has on later life disease risk and further research is required to develop the most effective preventative strategies related to it."

The authors point to some of the study limitations, such as relying on self-reported early life body size which may have affected the accuracy of their estimates.

Dr. Richardson adds: "It is remarkable that we were able to use human genetics data to separate how body size at different stages in life contributes towards disease risk. Future studies looking into the role that early life obesity plays in other types of disease, as well as investigating the biological mechanisms may help us further understand these findings. This includes using data on molecular traits, such as circulating metabolites and sex hormones, to evaluate whether they are involved as mediators between childhood obesity and later life disease. In doing so, we hope to develop a greater understanding of the most important biomarkers which will allow us to prevent—and even potentially to treat—disease."

More information: Use of genetic variation to separate the effects of early and later life adiposity on disease risk: mendelian randomisation study, *BMJ* (2020). DOI: 10.1136/bmj.m1203 , www.bmj.com/content/369/bmj.m1203

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